planations of the figures Dr. Leitch has produced. Either the newly born infant carries a reserve store of calcium sufficient to provide for adequate calcification during the months when the mother's milk does not provide enough—and it must be borne in mind that there is such a protective storage of iron—or the data of the metabolism experiments which Dr. Leitch has used for her analysis are misleading, perhaps because the milk was not of such a quality as to favour optimum calcium retention.

It remains uncertain in view of the evidence reviewed by Leitch whether changes in the dietary intake of calcium or phosphorus are reflected to any significant extent in the composition of a mother's milk. Our own results, admittedly scanty, suggest that such an effect is not detectable, at all events in the first two or three months of lactation. Some of the mothers we investigated had been receiving about 2 grammes of calcium lactate daily during the latter half of pregnancy, but this supplement to calcium-deficient diets did not appreciably influence the calcium content of their milks.

Conclusion

There is certainly need for more information, not only about the composition but also about the *yield* of milk from mothers whose diet, adequate in all other respects, provides at least 2 grammes of calcium, 1.5 grammes of phosphorus, and 300 I.U. of vitamin D daily. If such milk were made the subject of metabolic investigation on infants it is possible that some of the disturbing discrepancies would be dispelled.

Summary

The literature on vitamin D does not contain precise information as to the range of antirachitic potency of human milk.

The results of an analysis of human milks for vitamin D, calcium, and phosphorus are here recorded and an attempt made to correlate the vitamin D content of diet with that of the milk.

The conclusions derived from the analysis are given.

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THE PATHOLOGY OF CHLORIDE METABOLISM IN MAN*

BY

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During the past ten years considerable significance has been attached to disturbances of the metabolism of chlorides in man. The information gained from observations on patients and experimental work in animals has applications in diseases so diverse as intestinal obstruction, pyloric stenosis, chronic nephritis, oedema, obesity, hypertension, and Addison's disease. The principles which control the chloride balance of the body and determine the nature of the processes of absorption, secretion, re-absorption, and excretion of chloride are of importance in these diseases, and their significance is already reasonably well established. The phenomena which control the exchange of chlorides in tissue cells and fluids are not yet fully explained.

Normal Cycle of Chloride Metabolism

The average normal adult on an ordinary diet ingests about 12 grammes of chloride daily. This can be easily verified by estimating the daily excretion of chloride in the urine. In addition to the urinary excretion, small quantities of chloride are excreted in the faeces and limited amounts in the skin secretions (probably not more than 2 grammes daily in the absence of active perspiration). In forced sweating the skin loss in chloride may amount to 10 or 12 grammes daily (McCance, 1938). It should be borne in mind that clinical analogies to such abnormal skin losses occur in conditions where there is intense sweating, in acute febrile disturbances, in the sweating of tuberculosis and acute rheumatism, in pneumonia, in thyrotoxic crises, and in heavy muscular exertion in hot surroundings.

Apart from external losses by these routes—skin, urine, and faeces—there occurs a continual circulation of chloride to and from the intestinal lumen. The total quantities of chloride in circulation in this way daily in the normal person are somewhat indefinite. Approximate measurements of volume and chloride content of the various alimentary secretions give some basis for calculation, as shown in Table I (McCance, 1936).

TABLE I.—Intestinal Secretions: Chloride Content

Secretion	Vol. (c.cm.) in 24 Hours	Authority	Chloride Content (grammes)
Saliva	1,500	Bidder and Schmidt	7.5
Gastric juice ..	2,000-3,000	Nicol and Lyall	17
Bile	300-500	Pfaff and Balch	4
Pancreatic juice	500-800	Wohlegemuth	8
Succus entericus	3,000	Pregl	18
Total chloride			54.5

These figures are quite approximate, and probably form a conservative estimate.

Nicol and Lyall (1939a and 1939b), for example, as shown in Table II, were able to remove 29 grammes NaCl by gastric aspiration from a male patient in the first day, and a total of 93 grammes in six days' aspiration, the amount on the first day representing the uninfluenced gastric secretion of chloride in this individual.

* Read in opening a discussion in the Section of Physiology and Biochemistry at the Annual Meeting of the British Medical Association, Aberdeen, 1939.

TABLE II.—*Gastric Secretion : Chloride Content*

Date	Gastric Aspiration (c.cm.)	Chloride Content %	Total Chloride (Grammes)
12/9/38	4,260	0.680	29
13/9/38	2,320	0.707	17
14/9/38	2,080	0.628	13
15/9/38	1,690	0.533	9
16/9/38	1,800	0.595	11
17/9/38	2,070	0.641	14

Total chloride secreted : 93 grammes ; average, 15 grammes.

In a patient suffering from biliary fistula whom I have been able to investigate recently, the biliary flow showed an average volume of 1,000 c.cm., with chloride content of approximately 7 grammes daily, as shown in Table III.

TABLE III.—*Biliary Secretion : Chloride Content*

Date	Bile Vol. (c.cm.)	Chloride Content %	Total Chloride (Grammes)
24/4/39	780	0.70	5.5
25/4/39	540	0.73	4.0
26/4/39	480	0.69	3.3
27/4/39	720	0.73	5.3
28/4/39	1,200	0.73	8.9
29/4/39	930	0.70	6.5
1/5/39	1,080	0.72	7.8
2/5/39	1,380	0.66	9.2
3/5/39	1,020	0.70	8.5
4/5/39	1,320	0.70	9.3
6/5/39	1,320	0.69	9.2
8/5/39	1,200	0.70	8.5
Average	1,000	0.70	7.2

It would thus seem that about 50 to 60 grammes of sodium chloride is secreted into the alimentary canal in twenty-four hours, and the amount may be considerably greater in certain individuals. The actual rate of secretion is undoubtedly greater than 3 grammes hourly, and must take place relatively quickly during digestion and absorption of meals. This exchange would seem to make considerable demands on the available stores of chloride in the circulating blood and body fluids, an amount which different estimates place between 90 and 160 grammes for a man of 60 kilogrammes of body weight (Bartlett, Bingham, and Pederson, 1938).

We must assume that reabsorption occurs almost as rapidly as excretion, since no major reduction in the plasma chloride level was found by Dodds and Smith (1923) during gastric digestion. These workers found a fall of 10 to 15 mg. per 100 c.cm. in the plasma chloride during the first hour. In the second hour, while gastric secretion remained vigorous, the plasma chloride remained constant, and later showed a slight increase, to return to normal fasting level. The chloride secreted in the gastric contents is very rapidly reabsorbed by the intestine, and again forms part of the chloride store in the tissue fluids and blood plasma. The question of intestinal absorption of chloride is obviously rather a complicated physiological puzzle, since intestinal secretions contain up to 0.64 per cent. NaCl, while the chloride concentration of the intestinal contents for absorption is of the same order. The help of the physiologist is obviously required in the explanation of this anomaly.

Regulation of Chloride Content of Body Fluids

The regulation of the chloride content of the body fluids is dependent on the chloride content of the blood plasma. Most workers agree that this value remains between the limits of 580 and 620 mg. per 100 c.cm., values above 600 mg. per 100 c.cm. being found only occasionally. The chloride content of whole blood is considerably lower at about 440 mg. per 100 c.cm. on account of the low chloride value of the red blood cells. The exchange of chloride between blood plasma and body fluids forms one of the major means for stabilization of osmotic tension between those fluids. Table IV shows the average chloride content of the various body fluids.

TABLE IV.—*Chloride Content of Body Fluids*

Fluid	Chloride (as NaCl) mg. per 100 c.cm.
Blood plasma	600
Whole blood	438
Saliva	460
Gastric juice	670
Bile	700
Pancreatic juice	230
Jejunal juice	750
Ileal juice	400
Cerebrospinal fluid	730
Lymph	630
Milk	160
Sweat	350

The main controlling influences concerned in the maintenance of the chloride content of the blood plasma and body fluids are (a) the kidney itself and (b) adequate intake of chloride. Of these the former plays the major part, cessation of chloride excretion occurring as soon as the plasma chloride shows any tendency to diminution. So effective is this check against chloride loss that a minimal intake of chloride can be experienced for several days without any reduction of the plasma chloride level. The level of the plasma chloride at which renal excretion of chloride ceases is about 520 mg. per 100 c.cm. (Anderson and Lyall, 1937), as shown in two ways by Tables V and VI.

TABLE V.—*Case of Intestinal Obstruction : After Relief*

Time	Plasma Chloride mg. per 100 c.cm.	Urine Chloride mg. per 100 c.cm.
1 day	467	Nil
2 days	455	20
4 "	467	20
5 "	496	29
6 "	528	154
7 "	555	233
8 "	580	440

TABLE VI.—*Case of Gastric Aspiration*

Time	Plasma Chloride mg. per 100 c.cm.	Urine Chloride mg. per 100 c.cm.
1 day	606	310
2 days	599	204
3 "	526	20
4 "	467	21
5 "	410	10

This level of the renal threshold for NaCl is practically constant in health, although it may be altered in either direction in renal disease, with the kidney failing to pass chloride at levels above the usual renal threshold as in certain cases of renal disease with oedema, or allowing chloride to pass into the urine at values below 520 mg. per 100 c.cm. in some cases of intestinal obstruction and alkalosis with secondary toxic nephritis.

Requirements of Sodium Chloride

Although the average daily intake of sodium chloride is between 6 and 12 grammes, a large proportion of this amount is excreted daily. It seemed of importance, with regard to replacing the loss of chloride from the plasma and tissue fluids in hypochloraemia, to determine the actual basic requirements in chloride.

For this purpose healthy adults, during rest in bed, were placed upon a salt-poor diet and given additions of sodium chloride when chloride excretion had become constant. On mixed, ward diets the chloride excretion in these patients averaged 9 grammes daily. On restriction of the diet to 2.1 grammes of chloride daily the chloride excretion fell within four days to an average of 2 grammes, and remained at this level for three days. Upon the addition of 2 grammes NaCl to the diet the chloride excretion increased by 2 grammes, and upon a further addition of 2 grammes NaCl the chloride excretion increased to an average of 7 grammes. The basal requirement of chloride for this period of deprivation is therefore in the neighbourhood of 2 grammes daily while standard conditions are maintained and perspiration avoided (Falconer and Lyall, 1937a).

Results of Chloride Deficiency

McCance (1936) and his co-workers have made us familiar with the effect of experimental chloride deficiency in the normal adult.

By means of repeated sweating by radiant-heat baths and replacement of fluid by liberal water intake, they succeeded in causing a fall in the plasma chloride to 0.468 mg. per 100 c.cm., an increase in the blood urea to 81 mg. per 100 c.cm., with a reduction of sodium and chlorine of the blood plasma, an increase in the alkaline reserve, and increase in blood proteins. The symptoms experienced were severe, and included anorexia and nausea, lack of appetite, mental apathy, muscular cramps, a general sense of exhaustion, inability to undertake effort, and breathlessness and fatigue on exertion.

The symptoms described by McCance correspond very closely to those found in patients suffering from pathological salt deficiency, of which the commonest cause is pyloric stenosis with repeated and copious vomiting. The patient is drowsy, disoriented, and may be obstructive or violent. He is intensely dehydrated. Table VII shows the blood changes in a typical case, with return to normal values on improvement.

TABLE VII—Patient aged 66: Pyloric Stenosis

Date	Blood Urea mg. per 100 c.cm.	Plasma Chloride mg. per 100 c.cm.	Plasma CO ₂ vol. %	Plasma PO ₄ mg. %
5/12/32	162	408	118	3.1
7/12/32	166	438	92	5.3
8/12/32	150	461	92	4.3
9/12/32	140	480	88	4.0
10/12/32	116	496	—	3.4
12/12/32	114	584	80	—
13/12/32	104	560	72	2.6
14/12/32	78			
17/12/32	64			
3/1/33	35			

In certain instances, if it is found that fluids and saline cannot be replaced efficiently by intravenous medication, it may be necessary to carry out jejunostomy for this purpose (Falconer and Lyall, 1938).

In general, intestinal obstruction at lower levels than the pylorus may result in hypochloraemia, azotaemia,

alkalosis, and dehydration if the obstruction is in the upper part of the small intestine; or in azotaemia, dehydration with a variable disturbance of acid-base equilibrium, and no reduction in the plasma chloride (Falconer and Lyall, 1937b). Further variations are possible if fistula is included, as shown by Standard (1938). With duodenal fistula, dehydration and acidosis might actually be produced.

Gastric Chloride Secretion in Pathological States

Since the secretions of the gastric mucosa are reabsorbed normally in lower regions of the alimentary canal, the character of the gastric secretion in pathological states, and particularly during states of hypochloraemia, is of considerable importance in the preservation or restitution of the normal chloride content of the blood plasma and body fluids. Examination of samples of gastric contents taken at random from patients suffering from pyloric stenosis had suggested to us that the gastric mucosa could secrete a juice containing considerable quantities of chloride even when the plasma chloride level had fallen below normal. Suggestive confirmatory evidence that such was the case appeared in several publications.

McCance (1938) found that there was little change in the hydrochloric acid or total chloride content of the gastric juice during experimentally produced salt deficiency in man. Soley, Lagen, and Lockhart (1938) observed no significant alteration in the free acidity or total hydrochloric acid of the gastric juice during experimental hypochloraemia in three healthy men. Katsch and Mellinghoff (1933) removed quantities of NaCl up to 33 grammes in cases of peptic ulcer, with an average resultant reduction of 51 mg. per 100 c.cm. in the plasma chloride and an average fall of 20 per cent. in the volume of the gastric secretions, but without any appreciable change in the chloride concentration. Nicol and Lyall (1939a) investigated a series of patients suffering from pyloric stenosis and vomiting with severe hypochloraemia. Continuous sampling of the gastric contents over twenty-four-hour periods showed that the daily curves of free HCl, total HCl, and total chloride showed practically no difference from similar samples taken after return of the blood chemistry to normal (Table VIII).

TABLE VIII.—Average Values of Continuous Samples of Gastric Contents during Hypochloraemia and Period of Recovery

Case	Day	Plasma Chloride mg. per 100 c.cm.	Gastric Chloride mg. per 100 c.cm.	Free Acid	Urine Chloride mg. per 100 c.cm.
I	1st	350	628	26	10
	3rd	496	667	30	10
	10th	525	610	26	214
	11th	(gastro-enterostomy)			
	14th	526	635	28	310
	17th	570	680	34	298

The reverse procedure seemed to be necessary to establish this phenomenon with certainty.

For this purpose Nicol and Lyall (1939b) subjected two patients with pyloric stenosis and gastric hypersecretion, but without changes in the blood chemistry, to a similar experimental procedure. The gastric juices were withdrawn continually for periods up to six days when the blood chemistry showed the changes of gross alkalosis and hypochloraemia. Under these circumstances the gastric secretions showed little qualitative alteration, but decreased in volume (Table IX).

This evidence probably does not add much to the clarification of the vexed problem of the regulation of the gastric secretions, but it certainly seems to be clear that

TABLE IX

Case	Day	Gastric Juices			Blood		
		Vol. c.cm.	Chloride	Total Chloride (grammes)	Chloride mg. per 100 c.cm.	Urea mg. per 100 c.cm.	CO ₂ vol. %
1	1st	—	—	—	606	36	68
	2nd	4,260	0.707	30	599	40	84
	3rd	2,320	0.628	14.6	526	46	91
	4th	2,080	0.533	11	467	—	98
	5th	1,690	0.595	10	410	68	120
	6th	1,800	0.641	11.5	365	75	130
2	1st	—	—	—	540	28	55
	2nd	—	0.415	—	496	27	74
	3rd	2,560	0.450	11.5	—	26	84
	4th	3,020	0.420	12.7	468	36	96
	5th	2,110	0.452	9.5	345	43	95
	6th	1,010	0.397	4.0	336	54	102

the secretion of free HCl and chloride by the gastric mucosa is to a large extent independent of and uninfluenced by gross changes in the chloride content and alkaline reserve of the blood plasma. It is difficult to correlate these results with the findings of Apperly and Crabtree (1931) and Browne and Vineberg (1932), and it seems more probable that Gilman and Cowgill (1931, 1933) are nearer the truth when they suggest that the gastric mucosa shows a one-way permeability to chloride and that the chloride concentration of the gastric juice is such as to bring that secretion into osmotic equilibrium with the blood plasma.

Chloride Metabolism in Addison's Disease

During some of the observations already described on the chloride balance in cases of intestinal obstruction, the opportunity occurred to investigate two cases of Addison's disease.

In both the typical blood chemical changes found by Stewart and Rogoff (1925), Baumann and Kurland (1927), and others in adrenalectomized animals, and by Loeb (1933) and his associates in man, were present during the phases of Addisonian crises. One of these patients, however, had been under observation for several years, and although the presence of Addison's disease had been suspected the diagnosis could not be made with certainty until the recognized changes in the blood chemistry appeared. It was obvious that until this time no method was available by which the diagnosis of Addison's disease could be established with any degree of certainty in the early stages. Anderson and Lyall (1937) were struck by the great difference between the rate of urinary chloride excretion found in those cases, as shown in Table X, and in cases of intestinal obstruction with reduction of the plasma chloride levels.

TABLE X

Date		Plasma Chloride mg. per 100 c.cm.	Urine Chloride mg. per 100 c.cm.
28/2/37		534	390
29/2/37		531	360
1/3/37		511	290
5/3/37		496	700
6/3/37		488	880
7/3/37		496	470
8/3/37		479	310
		Injections of suprarenal	
9/3/37		452	270
11/3/37		438	200
12/3/37		502	210

It seemed to us that a method was available for the early diagnosis of Addison's disease from the ratio of the urinary chloride excretion to the plasma chloride level, and we stated that in the absence of evidence of diabetes or renal disease the finding of a chloride concentration in the urine of over, say, 200 mg. per 100 c.cm. when the plasma chloride level is below the minimal normal level of about 530 mg. per 100 c.cm. would strongly suggest the presence of Addison's disease.

Cutler, Power, and Wilder (1938) tested the validity of this suggestion by preliminary investigations on nine subjects with adrenal insufficiency and eight subjects without evidence of disease of the adrenal glands, and later by a standardized procedure in the examination of thirty-six additional subjects—eight with and twenty-eight without evidence of adrenal insufficiency. The test period consisted of three days on a diet containing 0.95 gramme of chloride ion, 0.59 gramme of sodium ion, and 4.1 grammes of potassium, with the addition of 0.033 gramme of potassium per kilogramme of body weight on the first and second days. Blood was drawn at 10 a.m. on the third day and urine collected from 8 a.m. to 12 noon on the third day. Determination of the sodium and chlorine content of the blood plasma and urine showed that "if the concentration of chloride in the specimen of urine on the third day exceeds 225 mg. per 100 c.cm., Addison's disease or adrenal insufficiency is strongly suggested; if it is less than 125 mg., adrenal insufficiency is made unlikely."

Confirmation of the findings of Cutler, Power, and Wilder have accumulated.

Dryerre (1939a) investigated four patients with Addison's disease, five suffering from asthenia not due to Addison's disease, and five normal controls. His results revealed that on the third day of the administration of a salt-poor high-potassium diet the concentration of the sodium in the urine is much higher in cases of adrenal insufficiency than in control subjects. Dryerre did not find the urinary chloride concentration in cases of Addison's disease to differ with such consistency from that of control cases. More recently Dryerre (1939b) has used diminution of the sodium concentration in the urine as an index of response to treatment by cortin and desoxycorticosterone in patients suffering from Addison's disease, and suggests that the method may be of value in standardizing the activity of preparations of cortical extract.

Within the past two years Anderson and I have been investigating patients with some of the signs or symptoms of Addison's disease—weakness, asthenia, pigmentation, hypertension, abdominal discomfort, loss of weight. Our technique differs from that of Cutler, Power, and Wilder, and is more simple.

The patient is given a low chloride diet for a period of five to six days. A shorter period may be necessary. The blood chemistry is investigated at the beginning and end of the period and the urinary chloride concentration compared with the plasma chloride level daily and at the end of the period. The normal ratio of plasma chloride to urinary chloride under these conditions is always greater than 3 to 1. In Addison's disease the urine chloride concentration approaches the blood chloride level, and where the ratio is below 2 to 1 a diagnosis of Addison's disease can be established with certainty.

Summary

This paper indicates the importance of disturbances of chloride metabolism in patients suffering from intestinal obstruction, pyloric stenosis, chronic nephritis, oedema, obesity, hypertension, and Addison's disease.

The exchanges of chloride in the alimentary canal are discussed, both normally and in pathological conditions.

Observations are made on the secretion of urinary chloride in hypochloraemic states, and the changes found in various types of hypochloraemia are illustrated.

It is pointed out that in Addison's disease the reduction in plasma chloride is associated with failure of the kidney to retain chloride.

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NOTES ON PSYCHIATRIC CASUALTIES OF THE FIRST DAYS OF WAR

BY

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Experience of the first two weeks of war indicates that the special stresses of that period have already produced psychiatric cases and problems of special types not usually met with in peace-time psychiatric practice. Though it may be considered premature, it is felt that a record of impressions of such cases should be made while they are yet in mind, particularly as they belong to a phase of the war which is now passed—a period when the much-discussed mass bombardment of civil populations in this country was considered, and appeared to be imminent, before evacuation was completed and before war morale was established.

Occurrence and Attributability to War Strain

A casualty hospital situated close to a great railway terminus in London is so placed as to receive patients who collapse in special circumstances. The glass-domed station, the crowds, loud-speakers, and confusion of curtailed rail services, together with evacuation of children and mothers, punctuated by air-raid-warning sirens, brought the war home to many as a reality for the first time. War morale had not yet been established by leadership, and a sense of security had not been generated by knowledge of what preparations had been made for the protection of the civilian population. In most of the cases the patient had lost touch temporarily with his or her family.

A striking contrast existed between newly precipitated cases and old patients who came up as usual to the psychiatric out-patients' clinic. These old patients were all asked how the present situation affected their symptoms or themselves. Typical replies were: "I don't take no account of that." "I'm afraid this illness makes me rather selfish. I hardly think of the war." A small

minority were troubled by fear of air raids, and it may be that others who failed to attend were those most affected.

The newly precipitated cases were brought in ambulances or by the police, having collapsed in the street or the station. On arrival they were still unconscious or confused. It was possible in all cases to restore consciousness and to obtain names and addresses, etc., in a very short time by reiterated reassurance and encouragement, continued until eyes opened and questions were answered. Typically, attention was apt to wander and irrelevant answers were given to questions. In one or two cases there was wild excitability, and this was usually controllable by quiet confident reassurance and persuasion. Only one true hysterical paralysis was seen. As the object of treatment was merely to enable patients to continue their journey out of London or to return them to their homes it has not been possible to follow subsequent developments in most of these cases. In one case a mother taking two children home from the seaside through London collapsed when she became cut off from them in the crowd. In this, as in other cases, there was a large element of fatigue, lack of sleep, and hunger. Alcohol played a part in a number of them, and there seemed to be an abnormal sensitivity to small quantities.

These cases were fairly equally distributed between the sexes. No children were included. They were of the less-educated classes. About twenty cases in all were seen, and the flow diminished markedly after the first week of war.

Cases simulating Acute Onset of Psychosis or of Severe Neurosis

Five cases fall into this class, and all but one were of the professional classes. There were three men and two women. They were doing responsible work and, except in one case, work directly connected with the state of emergency. There were other significant similarities. Four were intellectual types with a high sense of social responsibility; the state of stress had been established for a considerable time before the war broke out, and they had overworked, neglecting their own welfare for that of others, living on snacks and irregular meals and being unable to sleep adequately. In all the fear of a breakdown had been a factor for some time and had been compensated by increased press of activity, mental and physical. In all the onset was sudden and dramatic.

A man of 30 in a very responsible job calling for speed of decision and acuteness of judgment suddenly lost all energy, and after wandering aimlessly for an hour or so had a "black-out" of consciousness without collapsing. He was brought to hospital ten hours later with symptoms suggestive of paranoid schizophrenia.

A highly conscientious overworking governess of 28 had had sole charge of four small children in the country. She came up to London, losing some trunks on the way. The sight of sand-bagged buildings and the dark night streets seemed to alarm her unduly, and she suddenly said she must go out at once and find a job. She ran into the street and was eventually found in a police station. Two hours later, when seen at hospital, she was wildly frightened and suspicious, believing that a plan existed for her destruction in which the physician was a tool of the Government. She slept well with 15 grains of medinal, but next day was still more frightened and suspicious, and had to be placed in a mental hospital under an urgency order.

A medical man doing important organizing work on night duty had almost complete insomnia for a week and showed obsessional doubts and other obsessional symptoms.